News

Stress–Pollution Interactions: An Emerging Issue in Children's Health Research

Many of the diseases studied under the aegis of environmental health are multifactorial in nature. Investigators are now looking at psychological stress as a potential modifier of some children's response to certain environmental pollutants. But stress has been hard to measure and quantify, and the mechanisms by which it may affect physical health have been unclear. A small group of researchers is finding new ways to address these issues and explore stress–pollution interactions and their impacts on children's health (**p. A430**).

Advising Parents in the Face of Scientific Uncertainty: An Environmental Health Dilemma

Environmental health scientists are all too aware of the difficulty of making direct connections between exposures and individual health outcomes. Clinicians, meanwhile, are often reluctant to discuss potential threats for which there is no clear guidance. Yet many prospective and new parents are demanding information about environmental exposures that may affect their health and that of their children. Researchers and clinicians are collaborating to develop effective methods for communicating clearly and accurately with new and prospective parents about environmental health hazards—both what we know and what we don't know (**p. A436**).

Podcast

Early Influences on Mammary Gland Development, with Suzanne Fenton

Girls are developing breasts and going through puberty earlier than they did in the past. Now researchers are investigating the role environmental exposures may play in this trend and the potential long-term effects. In this podcast, host Ashley Ahearn discusses with researcher Suzanne Fenton how research on environmental exposures and mammary gland development in rodents might be used to assess chemical risks for humans.

Commentary

Reduction in New Jersey Air Pollution: 1950s–2010

Air pollution has been a topic of concern and study for hundreds of years. During the second half of the 20th century, the United States implemented regulations and controls to reduce the levels of criteria air pollutants and achieve the National Ambient Air Quality Standards (NAAQS) to protect human health, leading to decreases in the levels of toxic air pollutants. **Lioy and Georgopoulos (p. 1351)** trace the changes in air pollution in New Jersey to demonstrate the impact of local, state, and federal strategies to control emissions of pollutants and pollutant precursors from the 1950s until 2011. In New Jersey, the original NAAQS (1970–1995) have been achieved and significant progress has been made to meet revised standards for ozone and $PM_{2.5}$. Assuming that there is no change in current regulations, reductions in outdoor air pollution reductions should continue to address health risks associated with exposure to ozone and particulate matter and pollution. The authors conclude that future strategies to reduce air pollution need to consider biologically based susceptibility or age of populations, indoor air pollution, contents of pollutants in consumer and personal product, and fuels used to produce energy.

Reviews

Translating Arsenic Toxicology to Human Pathology

Although chronic arsenic exposure is a worldwide health problem, how arsenic exposure causes a variety of diseases is poorly understood. Research in this area is hampered because specific relationships between experimental and human exposures have not been established. **States et al. (p. 1356)** examined the use of phenotypic anchors to translate experimental data to human pathology and investigated research needs for which phenotypic anchors need to be developed. Disease outcome is likely dependent on cell-type–specific responses and interaction with individual genetics, other toxicants, and infectious agents. Potential phenotypic anchors include target tissue dosimetry, gene expression and epigenetic profiles, and tissue biomarkers. Translation to human populations requires more extensive profiling of human samples along with high-quality dosimetry. The authors conclude that anchoring results by gene expression and epigenetic profiling has great promise for data unification and that anchoring experimental results to specific human exposures will accelerate understanding of mechanisms of arsenic-induced human disease.

Academic–Community Partnership in Appalachia

Community engagement in research is strongly encouraged by the National Institutes of Health and the National Institute of Environmental Health Sciences, but there is little documentation as to how an academic–community partnership that implements community-based participatory research (CBPR) principles can be mobilized for research. **Haynes et al. (p. 1364)** created a bidirectional, academic–community partnership with an Appalachian-American community to identify the community's thoughts and perceptions about local air quality, its effect on health, and the perception of risk communication sources, with the goal of jointly developing and conducting an environmental health research project. The authors report that the quality and relevance of the study was enhanced because the community was engaged from the onset. The authors conclude that the principles of CBPR are useful in building a strong partnership and that academic researchers should consider working collaboratively with existing community-based risk communication sources.

Research

Heart Rate Variability and Cycling in Traffic

Few studies have examined the acute health effects of air pollution exposures experienced while cycling in traffic. **Weichenthal et al. (p. 1373)** conducted a crossover study in Ottawa, Ontario, Canada, to examine the relationship between traffic pollution and acute changes in heart rate variability in 42 healthy nonsmoking men and women who were not exposed to tobacco smoke in the home and did not take medications for preexisting cardiovascular or respiratory conditions. The authors also measured spirometry and exhaled nitric oxide. Participants cycled for 1 hr on high- and low-traffic routes, as well as indoors, with health measures collected before cycling and 1–4 hr after the start of cycling. Ultrafine particles (≤ 0.1 μm in aerodynamic diameter), $PM_{2.5}$, black carbon, and volatile organic compounds were measured along each cycling route, and ambient nitrogen dioxide and ozone levels were recorded from a fixed-site monitor. The authors conclude that short-term exposures to traffic pollution may contribute to altered autonomic modulation of the heart in the hours immediately after cycling.

Also see Science Selections, p. A443

Ambient Particulate Matter Activates NF-κB Signaling

Exposure to ambient air particulate matter (PM) increases rates of cardiopulmonary morbidity and mortality, but the underlying mechanisms are not well understood. **Silbajoris et al. (p. 1379)** examined signaling events involved in the expression of the inflammatory gene interleukin-8 (*IL-8*) in human airway epithelial cells (HAECs) exposed to ambient PM collected in an urban area of Mexico. The authors report that exposure resulted in a dose-dependent increase in *IL-8* mRNA expression relative to controls; PM exposure induced *IL-8* transcriptional activity in BEAS-2B cells that was dependent on the nuclear factor-kappa B (NF-κB) response element in the *IL-8* promoter. The authors conclude that their findings show that the increase in *IL-8* mRNA expression in HAECs exposed to PM_{10} is mediated through an NF-κB–dependent signaling mechanism, and that exposure to PM_{10} in ambient air can induce inflammatory responses by activating specific signaling mechanisms in HAECs.

Air Pollution and Cardiovascular Disease Biomarkers

Exposure to traffic-related air pollution is a risk factor for cardiovascular events, probably involving mechanisms of inflammation and coagulation. However, little is known about the relationship between cardiovascular outcomes and short exposures such as those experienced during commuting. **Zuurbier et al. (p. 1384)** studied 34 healthy adult volunteers who commuted for 2 hr by bus, car, or bicycle during the morning rush hour. The authors measured particle number, $PM_{2.5}$, PM_{10}, and soot exposure during the commute and estimated inhaled doses based on heart rate monitoring. Shortly

before and 6 hr after exposure, blood samples were taken and analyzed for Clara cell protein 16, blood cell count, and markers of coagulation and inflammation. The authors observed no consistent associations between air pollution exposures and the various biomarkers investigated. They concluded that air pollution exposure during commuting was not consistently associated with acute changes in inflammation markers, blood cell counts, or blood coagulation markers under the conditions used.

Biomass Smoke and Blood Pressure in Chinese Women

Almost half the world's population uses coal and biomass fuels for domestic energy. Limited evidence suggests that exposure to air pollutants from indoor biomass combustion may be associated with elevated blood pressure. **Baumgartner et al. (p. 1390)** assessed the relationship between air pollution exposure from indoor biomass combustion and blood pressure (BP) in women in rural China. They measured 24-hr personal integrated gravimetric exposure to fine particles ($PM_{2.5}$) and systolic BP (SBP) and diastolic BP (DBP) in the winter and summer among 280 women ≥ 25 years of age who lived in rural households using biomass fuels. Increases in $PM_{2.5}$ exposure were associated with increased SBP and DBP among all women. The authors conclude that $PM_{2.5}$ exposure from biomass combustion may be a risk factor for elevated BP and thus for cardiovascular events.

Also see Science Selections, p. A442

Phthalates, BPA, and Thyroid Hormones

Previous animal, *in vitro*, and human studies have suggested that exposure to phthalates or bisphenol A (BPA) may affect thyroid signaling. **Meeker and Ferguson (p. 1396)** explored the relationship between urinary concentrations of metabolites of di(2-ethylhexyl) phthalate (DEHP), dibutyl phthalate (DBP), and BPA with a panel of serum thyroid measures among a representative sample of 1,346 adults (ages ≥ 20 years) and 329 adolescents (ages 12–19 years) from the National Health and Nutrition Examination Survey (NHANES) 2007–2008. Among adults, the authors observed significant inverse relationships between urinary DEHP metabolites and total thyroxine (T_4), free T_4, total triiodothyronine (T_3), and thyroglobulin, and positive relationships with thyroid-stimulating hormone (TSH). Conversely, they observed significant positive relationships between DEHP metabolites and total T_3 among adolescents. Mono(3-carboxypropyl) phthalate, a secondary metabolite of both DBP and di-*n*-octyl phthalate, was associated with several thyroid measures in both age groups, whereas other DBP metabolites were not associated with thyroid measures. The authors conclude that their results support previous reports of associations between phthalates and altered thyroid hormones and suggest associations involving More detailed studies are needed to determine the temporal relationships and potential clinical and public health implications of these associations.

Mindin Regulates Environmental Airways Disease

Previous research as shown that the extracellular matrix protein mindin contributes to allergic airways disease, but the role of mindin in non-allergic airways disease has not been explored. To test the hypothesis that mindin (spondin 2, extracellular matrix protein; *Spon2*) would contribute to airway disease after inhalation of either lipopolysaccharide (LPS) or ozone, **Frush et al. (p. 1403)** exposed C57BL/6J and mindin$^{-/-}$ mice to aerosolized LPS , saline, ozone, or filtered air, and evaluated mice 4 hr after LPS/saline exposure or 24 hr after ozone/filtered air exposure. Physiological and biological responses were characterized by analysis of airway hyperresponsiveness (AHR), inflammatory cellular recruitment, total protein in bronchoalveolar lavage fluid, proinflammatory cytokine profiling, and *ex vivo* bronchial ring studies. The authors report that their results indicate that mindin modifies the airway response to both LPS and ozone. They conclude that their findings support a conserved role of mindin in production of proinflammatory cytokines and the development of AHR in two divergent models of reactive airways disease, as well as a role of mindin in contractility of airway smooth muscle after exposure to ozone.

Saharan Dust and Daily Mortality

Outbreaks of Saharan-Sahel dust over Euro-Mediterranean areas are often associated with exceedances of the European Union standard for PM_{10}. **Mallone et al. (p. 1409)** evaluated the presence of Saharan dust on the association between different particulate matter fractions and daily mortality in Rome, Italy. They studied 80,423 adult residents who died in Rome between 2001 and 2004 and performed a time-series analysis to explore the effects of different particle fractions on mortality. The authors report that increases in $PM_{2.5–10}$ and PM_{10} were associated with increased mortality due to natural, cardiac, cerebrovascular, and respiratory causes. Associations of $PM_{2.5–10}$ with cardiac mortality were stronger on Saharan dust days than on dust-free days; Saharan dust days also modified associations between PM_{10} and cardiac mortality. The authors report evidence of effects of $PM_{2.5–10}$ and PM_{10} on natural and cause-specific mortality, with stronger estimated effects on cardiac mortality during Saharan dust outbreaks. The authors conclude that additional research is needed to determine the toxicological and biological effects of PM from desert sources, and that potential health effects of dust from natural sources should be taken into account in setting air quality standards.

Peat Wildfire Smoke and Emergency Visits

The association of mortality and morbidity with exposure to urban air pollution is well established, but the possible health effects associated with exposure to wildfire emissions are not well understood. To investigate the association between cardiorespiratory outcomes in people exposed to haze and air pollution during a peat fire in eastern North Carolina in 2008, **Rappold et al. (p. 1415)** carried out a population-based study using emergency department (ED) visits reported through a syndromic surveillance program, NC DETECT (North Carolina Disease Event Tracking and Epidemiologic Collection Tool). They used aerosol optical depth measured by a satellite to determine a high-exposure window and distinguish counties most affected by the dense smoke plume from surrounding reference counties. In the exposed counties, they observed significant increases in cumulative relative risk for asthma, chronic obstructive pulmonary disease, and pneumonia and acute bronchitis. ED visits associated with cardiopulmonary symptoms and heart failure were also significantly increased. The authors conclude that the consistent increase in relative risk in the exposed counties for nearly all outcome categories during 5 days after exposure to wildfire smoke has potentially significant public health implications.

Pollution Properties and Hospital Admissions

Previous studies have provided evidence that health risks differ by air pollutant, but the results have generally varied by study and health outcome, raising concerns about effectiveness of many single-pollutant models to examine air pollution impacts. **Suh et al. (p. 1421)** categorized pollutants by chemical properties and examined their impacts on the odds of daily hospital admissions among a population of Medicare recipients > 64 years of age in metropolitan Atlanta, Georgia, for 1998–2006. Data were analyzed in two stages. First, the authors used a case-crossover analysis to simultaneously estimate effects of 65 pollutants on cause-specific hospital admissions, controlling for temperature and ozone. They then regressed pollutant-specific slopes from the first stage on pollutant properties. Results indicated that 24-hr transition metals and alkanes were associated with increased odds of hospital admissions for cardiovascular disease (CVD); transition metals were significantly associated with increased hospital admissions for ischemic heart disease, congestive heart failure, and atrial fibrillation; increased respiratory-related hospital admissions were significantly associated with exposure to alkanes; and aromatics and microcrystalline oxides were significantly associated with decreased CVD- and respiratory-related hospital admissions. The authors conclude that their two-stage approach provides new evidence that transition metals are consistently associated with increased odds of CVD-related hospital admissions.

CHILDREN'S HEALTH

Organochlorines, Thyroid Function, and Neurodevelopment

Exposure to organochlorine (OC) compounds can alter thyroid function in humans, and hypothyroidism during early life can adversely affect neurodevelopment in children. **Julvez et al. (p. 1429)** studied the relationship between developmental OC exposures and thyroid function and the relationship between thyroid function and subsequent neurodevelopment. The authors followed a population-based birth cohort of 182 children up to 5.5 years of age. The assessments included measuring concentrations of OC compounds in maternal pregnancy

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serum and milk, clinical thyroid parameters in maternal and cord serum, and subsequent neuropsychological outcomes of the children. Resin triiodothyronine uptake ratio (T3RU) was also assessed as an estimate of the amount of thyroxine (T_4)-binding globulin sites unsaturated by T_4: The T3RU is high in hyperthyroidism and low in hypothyroidism. The authors report finding inverse monotonic associations between OC exposure and T3RU, and positive associations between T3RU and better performance on most of the neuropsychological tests. The authors conclude that OC exposures may decrease the T3RU during early life and that minor decreases in thyroid function may be inversely associated with a child's neurodevelopment.

Prenatal Lead Exposure and Weight

Cumulative prenatal lead exposure, as measured by maternal bone lead burden, has been associated with smaller weight of offspring at birth and at 1 month of age, but whether this effect persists into early childhood is unknown. **Afeiche et al. (p. 1436)** investigated the association of perinatal maternal bone lead, a biomarker of cumulative prenatal lead exposure, with children's attained weight over time from birth to 5 years of age in a sample population consisting of three sequentially enrolled longitudinal birth cohorts recruited between 1994 and 2005 at maternity hospitals in Mexico City. Children were weighed at birth and at several intervals until 60 months of age, and maternal tibia and patella lead were measured at 1 month postpartum. The authors observed an association between increased maternal patella lead (not tibial lead) and decreased weight among females but not males, at 5 years of age. These associations were similar after controlling for concurrent blood lead levels between birth and 5 years. The authors conclude that other studies are needed to confirm their results.

PBDEs in Mexican and Mexican-American Children

Polybrominated diphenyl ethers (PBDE) have been found to be higher in residents of California than of other parts of the United States. **Eskenazi et al. (p. 1442)** investigated the role of immigration to California on PBDE levels in Latino children. The authors compared serum PBDE concentrations in a population of 7-year-old first-generation Mexican-American children born and raised in California [Center for the Health Assessment of Mothers and Children of Salinas (CHAMACOS) study] with 5-year-old Mexican children raised in the states in Mexico where most of the mothers had originated (Proyecto Mariposa study). PBDE serum concentrations in the California Mexican-American children were three times higher than their mothers' levels during pregnancy and seven times higher than concentrations in the children living in Mexico, and PBDE serum concentrations were higher in the Mexican-American children regardless of the length of time their mothers had resided in California or the duration of the child's breast-feeding. Data suggest that serum concentrations of PBDEs in these children resulted primarily from postnatal exposure. The authors conclude that levels of PBDEs observed in these Mexican-American children may present a significant public health challenge and that the health consequences of chemical replacements for PBDEs should be investigated and weighed against their purported fire safety benefits.

Also see Science Selections, p. A442

Climate Extremes and the Length of Gestation

Although more extreme heat conditions have been predicted for the future, there is little information available on the potential impact of such conditions on pregnancy length. **Dadvand et al. (p. 1449)** investigated the impact of maternal short-term exposure to extreme ambient heat on the length of pregnancy based on a cohort of 7,585 births that occurred during 2001–2005 in Barcelona, Spain. They applied three indicators of extreme heat conditions based on exposure to an unusually high heat–humidity index, and developed a two-stage analysis to quantify the change in pregnancy length after maternal exposure to extreme heat conditions, adjusting for a range of covariates. Extreme heat was associated with a reduction in the average gestational age of children delivered the next day, suggesting an immediate effect of this exposure on pregnant women. The authors conclude that further studies are required to confirm these findings in different settings.

Also see Science Selections, p. A443

PBDEs and Thyroid Hormones in Pregnant Women

Polybrominated diphenyl ethers (PBDEs), chemical additives used as flame retardants in commercial products, are bioaccumulative, persistent, and associated with several adverse health outcomes. **Stapleton et al. (p. 1454)** measured PBDEs and PBDE metabolites in serum collected from 140 pregnant women late in their third trimester who were recruited from within an ongoing observational prospective cohort study assessing the joint effect of social, environmental, and host factors on pregnancy outcomes [the Healthy Pregnancy, Healthy Baby (HPHB) Study]. The authors also sought to determine whether the PBDEs or their metabolites were associated with maternal thyroid hormones. Blood samples were collected from pregnant women during a routine prenatal clinic visit, and serum was analyzed for a suite of PBDEs, three phenolic metabolites, and five thyroid hormones. PBDEs were detected in all samples; two hydroxylated BDE congeners (4′-OH-BDE 49 and 6-OH-BDE 47) were detected in > 67% of the samples. BDEs 47, 99, and 100 were significantly and positively associated with free and total thyroxine levels, and with total triiodothyronine levels above the normal range. PBDEs and OH-BDEs are prevalent in this cohort, with levels similar to those in the general population. The authors conclude that PBDEs may be able to affect thyroid regulation throughout pregnancy; thus further research is needed to determine mechanisms through which PBDEs affect thyroid hormone levels in developing fetuses and newborn babies.

Micronucleus Frequencies in Umbilical Blood

The incidence of childhood cancer in Europe appears to be increasing by around 1% each year. Although the cause associated with this increase is not known, *in utero* and maternal exposure to genotoxic compounds from diet and the environment may be a risk factor for the development of cancer during childhood. **Vande Loock et al. (p. 1460)** investigated early genetic effects in newborns, and thus their potential risk of developing childhood cancer. The authors used the cytokinesis block micronucleus (CBMN) assay, a well-validated biomarker of cancer risk in adults, to determine micronucleus (MN) frequencies in both mononucleated (MONO) and binucleated (BN) T lymphocytes and to derive the cytokinesis block proliferation index (CBPI) in full-term and preterm newborns and their mothers. The authors note that results of multivariable analysis indicate the importance of considering gestational age when studying MN frequency in newborns. They also note the importance of assessing both MONO and BN T lymphocytes for biomonitoring of newborns, because the former reflects damage expressed during *in vivo* cell division and accumulated *in utero*, and the latter includes additional damage expressed as MN during the *in vitro* culture step. In addition, because of physiological differences and the age of circulating T lymphocytes, it is not clear whether MN frequencies in newborns can be interpreted in the same way as those in adults, or whether they are predictive for cancer, particularly childhood cancer.

Perfluorinated Compounds and ADHD

Toxicology studies have suggested that exposure to perfluorooctanoic acid (PFOA) and other perfluorinated compounds (PFCs) may be associated with alterations in human growth and development. **Stein and Savitz (p. 1466)** examined the association between serum PFC concentration and parent or self-report of doctor-diagnosed attention deficit/hyperactivity disorder (ADHD) with and without current ADHD medication. Of the children eligible for the study, 12.4% reported ADHD and 5.1% reported ADHD plus use of ADHD medication. The authors report that prevalence of ADHD plus medication increased with perfluorohexane sulfonate (PFHxS) levels. They also observed a modest association between perfluorooctane sulfonate and ADHD with medication. The authors note a decreased prevalence of ADHD at the highest exposure level but conclude that this may be a spurious finding related to the geographic determination of PFOA exposure in this population or to unmeasured behavioral or physiological correlates of exposure and outcome. Possible positive associations between other PFCs and ADHD, PFHxS in particular, warrant future investigation.

Traffic-Related Pollutants and Fe_{NO}

The fractional concentration of nitric oxide in exhaled air (Fe_{NO}) is a noninvasive marker of airway inflammation that has been associated with air pollution exposure. Several studies have examined the association of traffic-related pollutants (TRP) with Fe_{NO} in children, but results have not been conclusive. **Eckel et al. (p. 1472)** studied the association between Fe_{NO} in children and five classes of metrics of residential TRP: distances to freeways and major roads, including length of all and local roads within circular buffers around the home; traffic densities within buffers; annual average line source dispersion modeled nitrogen oxides (NO_x) from freeways and nonfreeway roads; and predicted annual average nitrogen oxide, nitrogen dioxide,

and NO_x from a model based on intracommunity sampling. In children with asthma, length of roads was positively associated with Fe_{NO}, with stronger associations in smaller buffers. Other TRP metrics were not significantly associated with Fe_{NO}. The authors conclude that length of road was the only indicator of residential TRP exposure associated with airway inflammation in children with asthma, as measured by Fe_{NO}.

Air Pollution and Respiratory Health in Nigeria

Association of childhood respiratory illness with traffic air pollution has been investigated largely in developed but not in developing countries, where pollution levels are often very high. **Mustapha et al. (p. 1478)** investigated associations between respiratory health and outdoor and indoor air pollution in schoolchildren 7–14 years of age in low-socioeconomic-status areas of the Niger Delta. Exposure to home outdoor and indoor air pollution was assessed by a self-report questionnaire. School air pollution exposures were assessed using traffic counts, distance of schools to major streets, and particulate matter and carbon monoxide measurements. The authors report that traffic disturbance at home was associated with wheeze, night cough, phlegm, and nose symptoms, whereas school exposure was associated with increased phlegm. Results suggest that traffic pollution is associated with respiratory symptoms in schoolchildren. The authors note that associations reported in other studies may have been underestimated because of nondifferential misclassification resulting from limitations in exposure measurement.

Microcystin and Childhood Liver Damage

Microcystin-producing *Microcystis* bloom is a severe water problem globally. Some reports indicate that chronic exposure to microcystin can result in liver damage in adults, but little information is available for children. **Li et al. (p. 1483)** measured microcystin concentrations in drinking water and aquatic food from two lakes and four wells in the Three Gorges Reservoir Region of China. Participants in the study were children 7–15 years of age (n = 1,322) who obtained drinking water from one of the tested sources, completed questionnaires, and provided blood samples for analysis of serum liver enzymes and microcystin. The authors detetected microcystin in most samples of water and aquatic food from the two lakes. Children who drank water from the lake containing the highest microcystin concentrations had a total estimated daily microcystin intake 5 times higher than the tolerable daily intake proposed by the World Health Organization for children. The authors report that hepatitis B virus (HBV) infection, use of hepatotoxic medicines, and microcystin exposure were associated with liver damage among the children in the study. The authors conclude that future research should investigate a more sensitive method and more appropriate biomarkers for detecting microcystin.

Impact of Introduced Chimney Stove on Newborn Birth Weight

A growing body of evidence suggests an association between exposure to household indoor air pollution from cooking fires and adverse neonatal outcomes such as low birth weight (LBW). **Thompson et al. (p. 1489)** examined the effect of reduced exposure to wood smoke during pregnancy on LBW of Guatemalan infants in the Randomized Exposure Study of Pollution Indoors and Respiratory Effects (RESPIRE). Pregnant women (n = 266) either received a chimney stove (intervention) or continued to cook over an open fire. Personal exposure to CO was used as an indicator of exposure to household air pollution and as a proxy for particulate matter exposures. Body weights were recorded for 174 eligible infants (69 born to mothers who used a chimney stove and 105 to mothers who used an open fire during pregnancy) within 48 hr of delivery. The authors report that women using chimney stoves had a significant reduction in mean exposure to carbon monoxide compared with those using open fires. On average, infants born to mothers who used a stove weighed more than infants whose mothers used open fires, and average birth weight was higher in infants born during the cold season compared with other infants. The authors conclude that use of a chimney stove reduced wood smoke exposures and was associated with reduced LBW occurrence, which is consistent with previous studies.

Prenatal Phthalate Exposure and Development at 6 Months

There are increasing concerns about the possible adverse effects of prenatal phthalate exposure on neurodevelopment of infants. As part of the Mothers and Children's Environmental Health Study (MOCEH), **Kim et al. (p. 1495)** studied the association between prenatal di(2-ethylhexyl) phthalate and dibutyl phthalate exposure and the Mental (MDI) and Psychomotor (PDI) Developmental indices of the Bayley Scales of Infant Development at 6 months of age. Prenatal mono(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP), mono(2-ethyl-5-oxohexyl) phthalate (MEOHP), and mono-*n*-butyl phthalate (MBP) were measured in mothers during the third trimester of pregnancy. The MDI was inversely associated with concentrations of MEHHP and MEOHP, and the PDI was inversely associated with MEHHP. In males, the MDI was inversely associated with MEHHP, MEOHP, and MBP; and the PDI was inversely associated with MEHHP, MEOHP, and MBP. No significant associations were observed for females. The authors conclude that prenatal exposure to phthalates may be inversely associated with the MDI and PDI of infants, particularly in males, at 6 months of age.

Manganese Exposure and Children's Behavior

Evidence of neurological, cognitive, and neuropsychological effects of manganese (Mn) exposure from drinking water (WMn) in children has generated public health concern. At elevated exposures, Mn has been associated with increased levels of externalizing behaviors, including irritability, aggression, and impulsivity, but little is known about potential effects at lower exposures, especially in children. Moreover, little is known about potential interactions between exposure to Mn and other metals, especially arsenic (As). **Khan et al. (p. 1501)** conducted a cross-sectional study of 201 children to investigate associations of Mn and As in tube well water with classroom behavior among elementary school children, 8–11 years of age, in Araihazar, Bangladesh. Data on exposures and behavioral outcomes were collected from the participants at the baseline of an ongoing longitudinal study of child intelligence, and study children were rated by their schoolteachers on externalizing and internalizing items of classroom behavior using the standardized Child Behavior Checklist-Teacher's Report Form (TRF). The authors report that WMn was positively and significantly associated with TRF internalizing, TRF externalizing, and TRF total scores. They also observed a positive monotonic dose–response relationship between WMn and TRF externalizing and TRF total scores among the study participants**.** These observations reinforce the growing concern about the possible neurodevelopmetnal effects of WMn in children.

Acute Respiratory Inflammation and Black Carbon

Lin et al. (p. 1507) estimated associations and exposure–response relationships between acute respiratory inflammation in schoolchildren and black carbon (BC) and $PM_{2.5}$ concentrations in ambient air before and during the air pollution intervention for the 2008 Beijing Olympics. Exhaled nitric oxide (eNO) was measured as a biomarker of acute respiratory inflammation, and hourly mean air pollutant concentrations were used to estimate BC and $PM_{2.5}$ exposure. The authors report that air pollution concentrations and eNO were clearly lower during the 2008 Olympics. BC and $PM_{2.5}$ concentrations averaged over 0–24 hr were strongly associated with eNO. In a two-pollutant model, estimated effects of BC were robust, but associations between $PM_{2.5}$ and eNO decreased with adjustment for BC. eNO was also associated with increases in hourly BC concentrations up to 10 hr after exposure, consistent with effects primarily in the first hours after exposure. The authors conclude that recent exposure to BC was associated with acute respiratory inflammation in school children in Beijing and that lower air pollution levels during the 2008 Olympics were associated with reduced eNO.

Aviation Gasoline and Childhood Blood Lead Levels

Aviation gasoline, commonly referred to as avgas, is a leaded fuel used in small aircraft. Recent concern about the effects of lead emissions from airplanes has motivated the U.S. Environmental Protection Agency to consider regulating leaded avgas. **Miranda et al. (p. 1513)** investigated the relationship between lead from avgas and blood lead levels in children living in areas surrounding airports, where elevated concentrations of ead from avgas may be present in air and potentially deposited to soil. The authors used regression analysis to examine the relationship between residential proximity to airports and blood lead surveillance data in children 9 months to 7 years of age. Results suggest that children living within 500 m of an airport where planes use leaded avgas have higher blood lead levels than other children, with some effect evident among children living within 1,000 m of airports. The authors conclude that their results are directly relevant to the policy debate regarding the regulation of leaded avgas.